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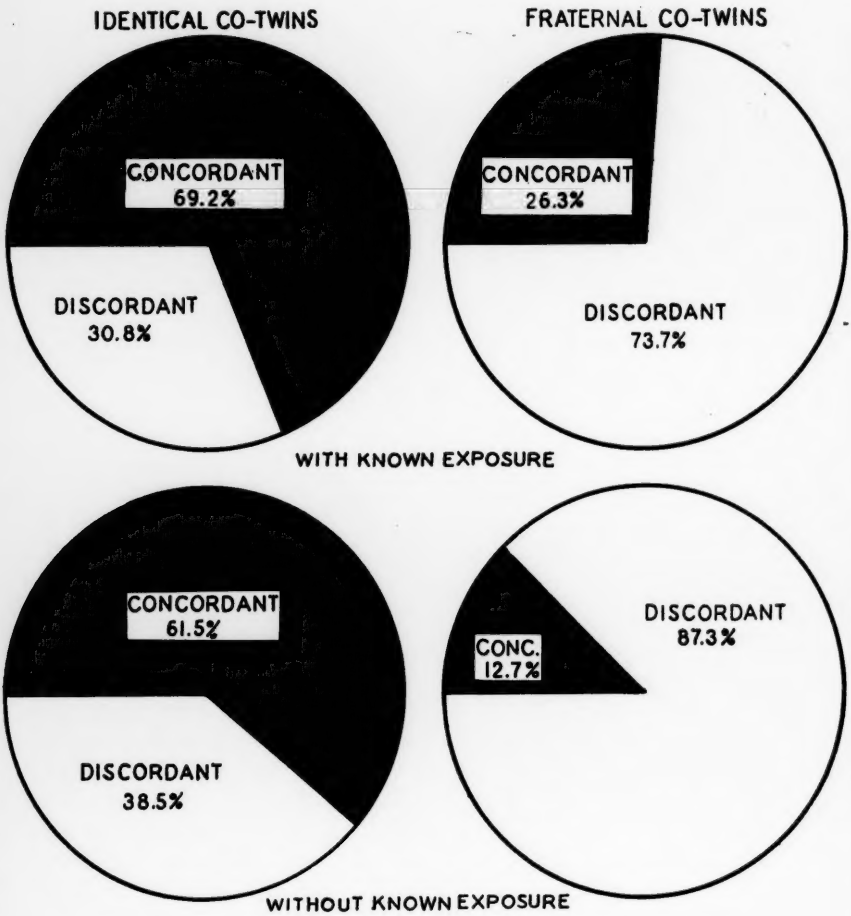

HISTORY OF TWINS ONE OF WHOM HAS TUBERCULOSIS
(SEE PAGE 296)

Gene-Chemistry in Clover

Amnesic Agronomy

VOLUME 34 OCTOBER, 1943 NUMBER 10

The American Genetic Association

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The

Journal of Heredity

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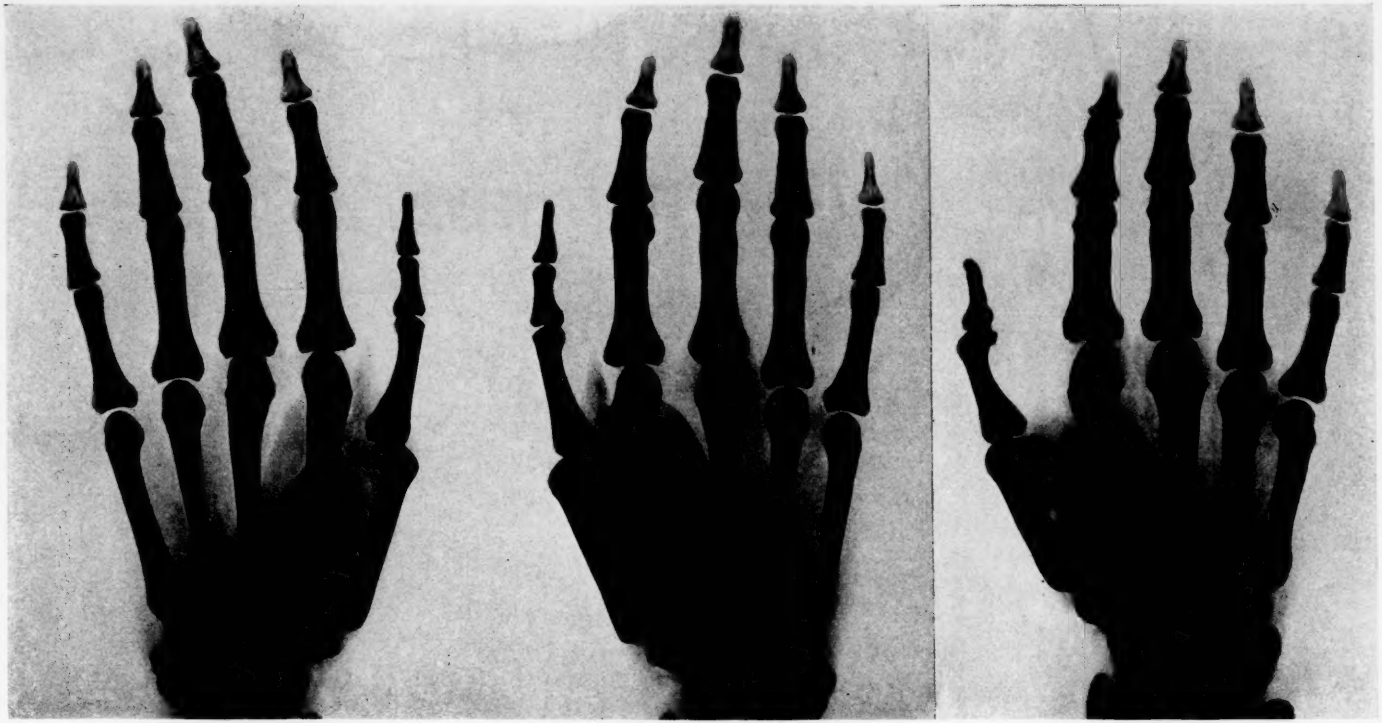


FIFTH FINGERS INSTEAD OF THUMBS

Figure 1

X-ray photographs of hands of a member (V-7 in Figure 2) of family having an extra phalange in the thumb so that these digits are essentially "fifth fingers." At the right is shown a normal thumb with but two phalanges. The thumbs of both hands of all affected individuals have this characteristic. There were no other obvious departures from normal development.

HEREDITARY HYPERPHALANGISM OF THE THUMB

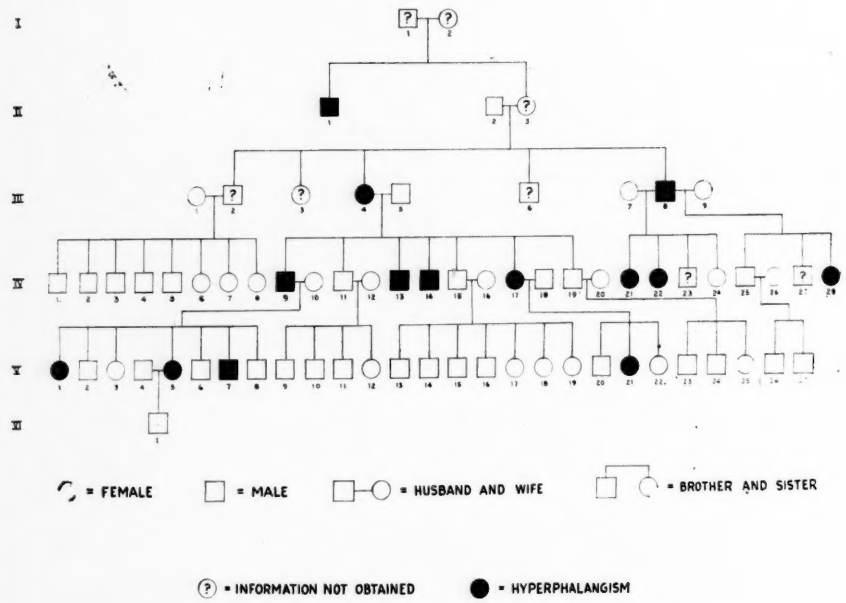
E. ROBERTS
University of Illinois

SEVERAL cases of abnormalities of the thumb have been reported. A case of short thumbs caused by shortened phalanges and inherited as an autosomal Mendelian dominant has been described by Breitenbecher;[2] a shortened condition caused by a short terminal phalanx occurring in one or both thumbs, also apparently an autosomal dominant, by Hefner[5]; and brachyphalangism by Hoffman.[7] Two cases of supernumerary distal phalanges have been reported by Reeves[11] and Weingrow.[13] Hyperphalangism of the fingers but not of thumbs appeared in a family with brachydactyly recorded by Unterrichter.[12] Hefner[6] described a case of hereditary polydactyly associated with extra phalanges in the thumb. In this case polydactyly occurred in both hands and feet and the thumbs with three phalanges were crooked. Kirmisson[8] described a case in which the thumbs had three phalanges but were crooked and malformed. X-ray examination indicated that anatomically these were thumbs and not duplications of fingers as might probably occur. Kristjansen[9] described a case of thumbs with three phalanges, wherein the hands were abnormal in appearance. Manoiloff[10] reported a case of polydactylism with six digits on hands instead of five. He concluded that both "thumbs" must be considered as supplementary fingers connected with not one but two metacarpal bones. Windle[14] described several cases reported to be hereditary in which some individuals had three phalanges in both thumbs while others had only one three-jointed thumb. Grynkraut[4] reported a case of three phalanges in an extra digit in which he suggested as a result of X-ray studies the possibility of absence of the thumb and duplication of an index finger. Arquembourg[1] and Frere[3] described thumbs with three phalanges. In the latter case enough information was given to indicate the peculiarity was inherited as a dominant.

A student in one of my classes called attention to the three-jointed condition of his thumbs, a peculiarity he said was found in several members of his family. Fairly complete information was obtained of four generations, the results of this survey being given in the family pedigree (Figure 2). An X-ray photograph of the hands of the propositus (V-7) shows three well formed phalanges in both thumbs (Figure 1); for comparison the right hand of a person with a normal two-phalange thumb is also shown. As far as could be ascertained, the digits of the feet and other digits of the hands were normal. Apparently this condition is inherited as a simple dominant character. In no case where the information is complete does the condition appear in a son or daughter unless it is present in one of the parents. In generation I (see Figure 2) information was not available, as was also the case of II-3. Based on results in later generations, probably either II-1 or II-2 and III-3 had this condition. In cases where one parent showed the extra phalanx of the thumb, information was obtained on twenty-three sons and daughters. Among these, twelve were normal and eleven had three-jointed thumbs. In generation III, the affected individual III-8 had children by two normal wives. Hyperphalangism occurred in children from both marriages. None of sixteen children from parents known to be normal was found to show this condition.

Summary

A pedigree of a family in which hereditary hyperphalangism of the thumbs is given. This condition is apparently dependent upon an autosomal dominant



FOUR GENERATIONS OF EXTRA-JOINT THUMBS

Figure 2

For three generations the extra-jointed thumbs have been inherited as a simple dominant.

gene. In this case hyperphalangism was confined to the thumbs of both hands was not accompanied as far as could be ascertained by any other abnormal characteristics.

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TABLE I. Occurrence of hyperphalangism of thumbs in families in Figure 2.

Parents	Children			
	Normal		Hyperphalangism	
	Sons	Daughters	Sons	Daughters
Father-hyperphalangism Mother-normal	4	2	2	5
Father-normal Mother-hyperphalangism	5	1	3	1
Father-normal Mother-normal	11	5	-	-

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TWIN STUDIES ON GENETIC VARIATIONS IN RESISTANCE TO TUBERCULOSIS

FRANZ J. KALLMANN AND DAVID REISNER

(Concluded from the September issue)

III. Statistical Data on Reinfection Tuberculosis

THE material reported in this study for analyzing the frequency of tuberculosis among the relatives of twin index cases affected by reinfection tuberculosis consists of 308 complete families. In addition to 616 twin partners, whose age and sex distributions were shown in Table I, there is an unselected series of 930 full siblings, 74 half-siblings, 688 parents, and 226 marriage partners of twin patients, making a total of 2,534 persons who have been investigated with respect to tuberculosis and contact conditions.

The collective morbidity rates for all relationship groups included in the survey are compared in Table IV. As regards the uncorrected rates for the siblings and half-siblings it may be noted that they relate to all individuals above the age of fourteen, that is, to 720 siblings and 42 half-siblings who are fifteen years or older. This statistical procedure seems advisable, since the age group 0-14 is not represented in any of the other relationship groups of this survey. When all 930 siblings and 74 half-siblings are used as rates of reference, their collective morbidity figures amount to 13.5 and 5.4 per cent, respectively.

Regardless of whether the uncorrected or corrected rates are taken into account, they clearly indicate that the chance of developing manifest tuberculosis increases in strict proportion to the degree of blood relationship to a tuberculous index case. The respective rates amount to 6.2 and 7.1 per cent in the marriage partners; 9.5 and 11.9 per cent in the half-siblings; 16.6 and 16.9 per cent in the parents; 18.9 and 25.5 per cent in the full siblings; 18.3 and 25.6 per cent in the dizygotic co-twins. The highest morbidity figures (61.5 and 87.3 per cent, respectively) are found in the monozygotic twin partners, that is, in the category of consanguinity implying complete homozygosis* for the given genotype. Their corrected concordance rate expresses the chance for a person above the age of 29, who is genetically identical with a tuberculous index case and has been exposed to infection, to develop manifest tuberculosis (Figure 3).

The correlation between consanguinity and frequency of tuberculosis is so consistent that it does not seem possible to explain all these group differences in morbidity merely by a more intimate contact between closer relatives. The sibling figure is about eighteen times higher than the expected general average frequency and also exceeds, possibly in part for reasons of selection with respect to the parents, the respective rates for the half-siblings, parents and marriage partners. The concordance rate for the dizygotic twins corresponds closely with the morbidity rate for the full siblings and shows a distinct difference only from the rates for the other sibship groups. The morbidity figure for the husbands and wives of the twin patients is about midway between the rates for the general population and the half-siblings, although their contact with the index cases may be assumed to be far more intimate than that of the half-siblings. In fact, exposure alone may not account for the total difference between general average expectation and the morbidity rate for the marriage partners, since marriage to a tuberculous patient statistically represents a definite selective factor with reference to the probability of being affected by the same disease.

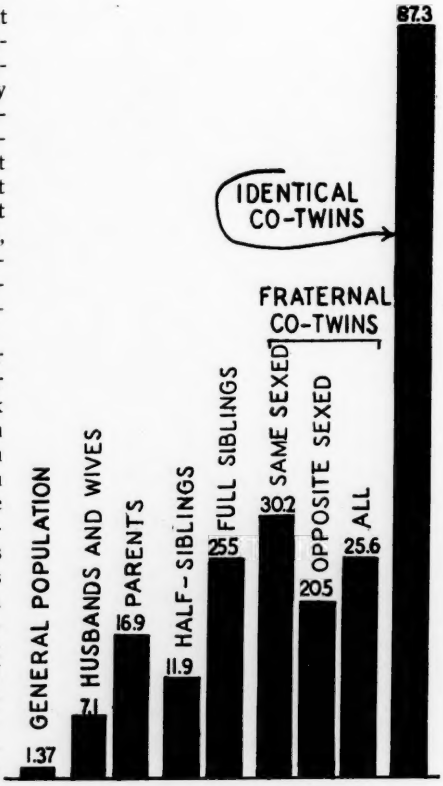
*A person is homozygous with respect to a genetic factor, if the particular gene is inherited from both parents so that the two corresponding alleles are alike in this person.

In order to exclude the possibility that the marked difference in morbidity between monozygotic and dizygotic co-twins might be sufficiently explained by factors other than genetic, a more detailed analysis of the index cases was undertaken. It is shown in Table V that the two groups of index cases do not differ significantly in the average age at disease onset, the frequency of exposure, or the ancestral taint conditions with respect to the known incidence of tuberculosis in the preceding generations [family history of tuberculosis].

No history of exposure to another source case has been ascertained in either group for about one-half of the index cases (52.6-55.2 per cent). However, in 44.6 per cent of all non-exposed twin patients it is known that there have been additional cases of tuberculosis in the direct or collateral lines of ancestry without personal contact between the twins and their affected blood relatives. This observation is definitely in favor of a genetic theory, particularly since it cannot be taken as absolutely certain that the family history in the other half of these index cases was really free of tuberculosis.

Concerning the frequency of tuberculosis in the twin partners of the index cases it is revealed in Tables VI-VIII that the collective morbidity rates for the co-twins are modified by a variety of secondary factors, genetic as well as non-genetic, but certainly not to an extent which would explain the marked difference between the two types of twins. In fact, regardless of whether these modifications are associated with race, sex, age, or parental taint, they are of similar extent in the groups of the siblings and dizygotic co-twins, but play only a minor role in the monozygotic group, thereby emphasizing the genetic nature of the factors for deficient resistance in this category.

It is shown in Table VI that the morbidity expectation among the siblings and dizygotic twin partners is about twice as high for the descendants of tuberculous parents as for the descendants of non-tuberculous parents, while this

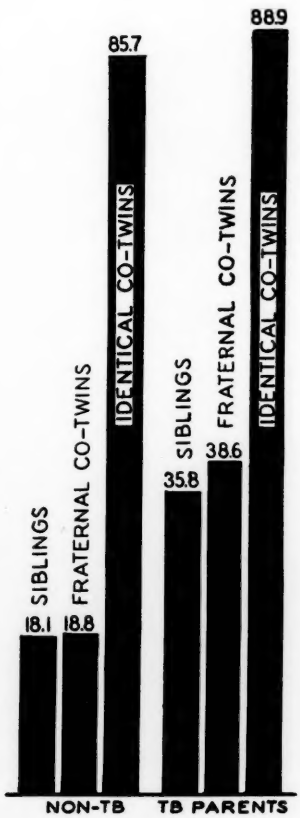


TUBERCULOSIS MORBIDITY RATES OF RELATIVES OF TWIN INDEX CASES

Figure 3

This chart compares the corrected total tuberculosis morbidity expectation, as a percentage, in the general population with the rate for various categories of relatives (over 14 years old) of twin index cases. When an identical twin develops manifest tuberculosis, the chances for the development of manifest tuberculosis in his co-twin will be nearly three times greater than when the index case is a same-sexed fraternal twin.

difference practically disappears in the one-egg twin partners. Their morbidity rates are unaffected by the possibility of being homozygous for deficient resistance because of having or not having a tuberculous parent. Instead, their chance of developing the disease appears largely determined by the genetic constitution of monozygotic twin partners of tuber-



EFFECT OF TUBERCULOSIS IN PARENTS ON RATES IN CO-TWINS

Figure 4

When one or both parents of an index case are known to have been tuberculous, the morbidity rate for the siblings and fraternal co-twins will be about twice as high as it is when neither parent has had the disease. The much higher rate for identical co-twins remains about the same regardless of whether the parents were tuberculous or not.

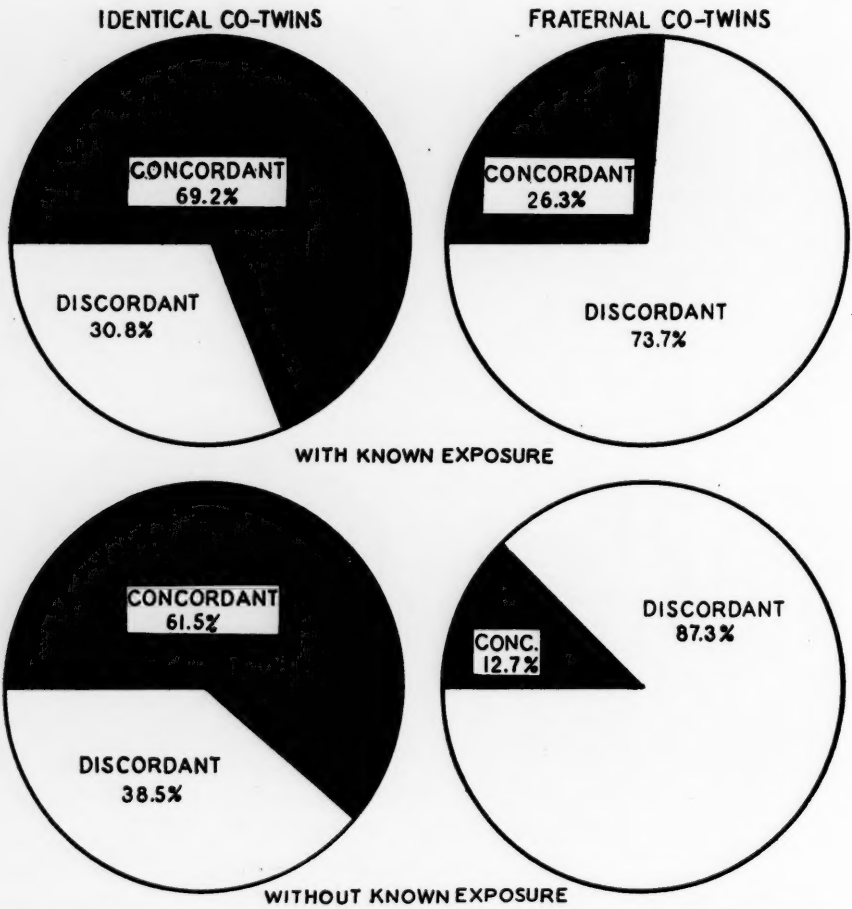
culous index cases (Figure 4).

With respect to the marked differences in morbidity as found between the sibships with and without tuberculous parents in the groups of the siblings and dizygotic twins, two possible explanations may be considered to account for the increased morbidity in the sibship groups with tuberculous parents. The increase may be the result of either intensified exposure or of the greater statistical chance of inheriting a deficient genotype from a homozygous parent. However, since the factor of additional exposure does not show a significant effect upon the concordance rate of the monozygotic twin partners, it does not seem sufficient to explain the variations in morbidity in the other sibship groups. In fact, if intensified exposure were the main cause of these differences, it would be difficult to understand why the dizygotic twin partners who had a tuberculous parent in addition to a tuberculous co-twin do not show a higher morbidity rate than the monozygotic twin partners without a tuberculous parent.

Variation in concordance between opposite-sexed and same-sexed pairs cannot exist in the monozygotic twins and is present only to a limited extent in the siblings and dizygotic twins. The range of the former group is from 21.7 to 27.7 per cent, and that of the latter group from 20.5 to 30.2 per cent (Table VI). These discrepancies are an indication that fraternals belonging to the same sex have a greater chance of being alike in those specific and non-specific conditions which favor the development of tuberculosis. It is evident, however, that the given sex variations are by no means extensive enough to permit a non-genetic explanation for the entire difference, or a major part of the difference, between the concordance rates of one-egg and two-egg twin pairs.

The excess of female over male morbidity rates in each of the sibship groups is definitely in accordance with statistical expectation. Most of the twin patients and their siblings were between the ages of fifteen and twenty-nine when they developed the disease, which means they were still in those age groups in which the general mortality rate of the female is also found to exceed that of the male.

A similar sex variation in morbidity reveals itself in the finding that the brothers and male twins of the index cases had a higher average age at disease onset than the sisters and female twins (Table VII). This sex difference amounts to 5.4 years in the siblings and



CONCORDANCE IN CO-TWINS OF INDEX CASES WITH AND WITHOUT KNOWN EXPOSURE TO TUBERCULOSIS

Figure 5

Interaction of genetic and environmental exposure factors is illustrated by these four comparisons between identical and fraternal co-twins of index cases. The existence of a known source of exposure increases the concordance of fraternal co-twins to a statistically significant extent, but does not increase significantly the concordance of identical co-twins.

to 2.6 and 5.5 years, respectively, in the monozygotic and dizygotic twin groups, while the total age differences between the three sibship groups are only of limited extent. Statistically it is to be expected, of course, that the average age at disease onset will be somewhat lower in the siblings than in the twin partners of tuberculous index cases, since only the siblings have an equal chance of being older or younger at disease onset than the index cases.

It is also shown in Table VII that the variations in concordance between the two types of twins are not explainable on the basis of differences in the period of time during which the twin partners were under observation before they were

classified as concordant or discordant. The monozygotic as well as the dizygotic members of the discordant twin groups had already reached an average age of thirty-one years at the time of their examination for this survey, and both categories of co-twins had at that time been discordant for about six years since the development of tuberculosis in the index cases.

The distribution of the sibship morbidity rates according to fatal and non-fatal courses of the disease is demonstrated in Table VIII. In the sibling group, this table also takes into account the variations in morbidity and fatality rates according to race (white and non-white persons) and according to whether these persons are related to fatal or non-fatal index cases. This differentiation has been limited to the full siblings, since the corrected morbidity and fatality figures for the other sibship groups are not based on rates of reference which would safely allow several further subdivisions.

The most interesting finding of this analysis is that there is no significant variation between white and non-white sibships with regard to their total morbidity figures. Fatal and non-fatal cases are about equally distributed in all the four sibship groups. The main effect of the racial factor reveals itself in a marked excess of fatal over non-fatal cases in the non-white group, which shows a fatality rate of 22.9 per cent as compared with 10.8 per cent for the white group. It may also be noted that the morbidity and fatality figures for those siblings who are related only to fatal index cases do not significantly differ from the respective rates for the total number of siblings.

Additional evidence against a simple correlation between closeness of blood relationship and increasing similarity in environment with correspondingly intensified opportunity for infection is obtained by an investigation of the distribution of exposed and non-exposed co-twins in the two groups of twins (Table IX). This analysis reveals that 20.5 per cent of all monozygotic pairs are concordant *without known exposure* of the co-twins, while 56.1 per cent of all dizygotic twin partners are discordant *despite known exposure* either to an index case or to another family member. One may also note that 61.5 per cent of the monozygotic pairs with non-exposed co-twins are concordant although these twin partners had no history of known exposure, and that 73.7 per cent of the exposed dizygotic twin partners are discordant in spite of known exposure.

The significance of intimate contact may or may not find its adequate expression in the statistical differences from 69.2 to 61.5 per cent and from 73.7 to 87.3 per cent, respectively, as found with respect to exposure between the concordant twin partners of the monozygotic group and between the discordant twin partners of the dizygotic group. It may also be important in this connection that the increase in the rate of concordant co-twins due to the factor of exposure is greater in the dizygotic group (12.7 to 26.3 per cent) than in the monozygotic group (61.5 to 69.2 per cent). There can be no doubt, however, that any such variation in relation to exposure does not suffice to explain a ratio of 1:3.5, or 25.6:87.3 per cent, as has been obtained for the collective morbidity rates of dizygotic and monozygotic twin partners (Figure 3).

That a genetic mechanism modifies individual resistance to tuberculosis is demonstrated still more clearly if the similarities in extent and outcome of the disease are taken as additional criteria of comparison. This is the objective of Table X* which compares the co-twin groups with similar and dissimilar behavior (satisfactory and unsatisfactory

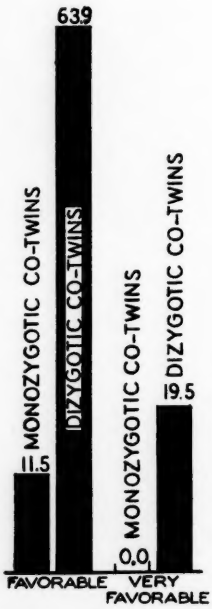
*The authors take pleasure in acknowledging the assistance rendered by Miss Jean Downes of the Milbank Memorial Fund and Dr. Joseph Zubin of the New York State Psychiatric Institute and Hospital in tabulating the material of Tables X and XI. They also wish to express their appreciation of the generous help extended by Drs. Lowell J. Reed and Paul R. David of Johns Hopkins University, Department of Biostatistics, in the statistical analysis of the entire survey.

resistance) to the disease, instead of comparing the twin groups with and without manifest tuberculosis as was done by the use of the concordance and discordance rates. The group designated as sufficiently resistant includes the sub-groups II and I, that is, the co-twins who developed either mild forms of tuberculosis with subsequent arrest of the disease or no tuberculosis at all in spite of definite exposure; while the insufficiently resistant group consists of the far advanced and fatal cases of tuberculosis as classified in the sub-groups III and IV.

In comparing dissimilarity and similarity in behavior to the disease, one obtains a ratio of 2:16 (9:69) in the monozygotic group, and a ratio of 2:1 (147:83) in the dizygotic group. In other words, similar behavior is about eight times more frequent than dissimilar behavior in monozygotic twins, but it is only one-half as frequent in dizygotic twin partners. Consequently, the difference in similarity of resistance between the two twin groups is expressed by a ratio of 1:16, which far exceeds the difference found in their original morbidity rates.

In fact, the chance of dying from tuberculosis is practically zero for a tuberculous patient who is the monozygotic twin of, or genetically identical with, a person who remains free of tuberculosis despite definite exposure to infection. However, the chance of dying is 1:4, if the person is merely the patient's sibling or dizygotic twin, which means that he is as likely to differ in the inherited elements for a satisfactory resistance as are two brothers or sisters.

Table XI expresses the same data in rates rather than in ratios, classifying satisfactory resistance to tuberculosis according to favorable and very favorable forms of resistance. In the monozygotic group, about 12 out of 100 twin partners of tuberculous index cases showed favorable resistance and none showed very favorable resistance, if their co-twins were insufficiently resistant. In the dizygotic group, however, favorable resistance was seen in 64 out of 100 twin partners of insufficiently resistant index cases, and very favorable resistance in about 20 out of 100 partners (Fig. 6).



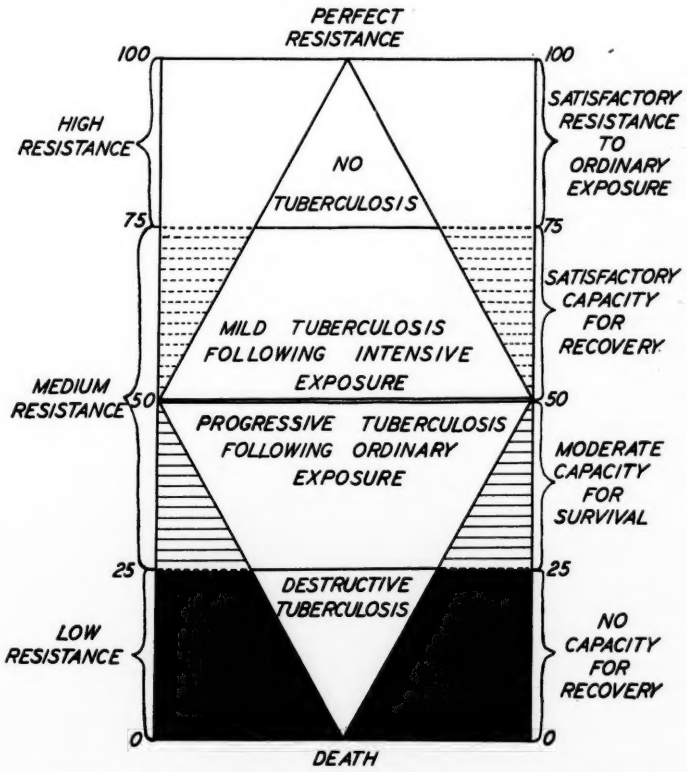
CHANCES OF SATISFACTORY RESISTANCE OF CO-TWINS

Figure 6

The chances that the co-twins of insufficiently resistant index cases will have satisfactory resistance to the disease are compared for identical and fraternal twins.

These findings demonstrate the existence of inherited differences in the individual vulnerability to tuberculous disease and seem best interpreted as a result of a graded protective character based on multi-factorial inheritance. The possible range of variations in genetically determined resistance factors is schematically illustrated in Figure 7. The range apparently extends from a highly resistant type which develops no manifest tuberculosis under ordinary conditions of exposure; through the most common type showing varying intermediary degrees of resistance; to a nonresistant type characterized by rapidly destructive tuberculosis.

It may not be without significance that there is a considerable discrepancy



RANGE OF VARIATION IN RESISTANCE TO TUBERCULOSIS

Figure 7

This chart illustrates the graded variations of inherited resistance factors in relation to exposure and the final outcome of the disease. A highly resistant person will develop no tuberculosis under ordinary conditions of exposure, while a nonresistant person will tend to develop progressive disease whenever exposed to infection. It is the middle group with medium resistance, including about half of a given population, in which therapy and prevention may be expected to offer the relatively best returns.

between the ratio of 1:16 as found for the dissimilarities between the two groups of twins in the extent of their resistance to progressive disease, and the ratio of 1:3.5 as found for their differences in resistance to any form of clinical tuberculosis. This finding seems to be in line with the previously offered suggestion that the factors for resistance to the invasion of the tubercle bacillus and the factors for resistance to the spread of the established disease should not be assumed to be entirely identical with each other. In order to clarify this complex genetic problem more satisfactorily, however, it will be necessary to identify the morphological basis for the various constitutional mechanisms which have been shown in the present study to be capable of modifying susceptibility or resistance to tuberculosis.

Such an analysis in turn requires the evaluation of all the clinical and anthropometric data which will be discussed in the subsequent parts of this report. Meanwhile, a number of general conclusions may be sufficient to summarize this first section of the inquiry into the

heredo-constitutional variability of tuberculosis.

Summary

1. An investigation of the possible significance of genetic factors in the development of tuberculosis was undertaken by means of the "Twin Family Method." This method combines the technique of the original "twin method," with the principles of a family study including the other sibship groups as well as the spouses of the twin index cases.

2. The study was organized with the cooperation of the tuberculosis hospitals

TABLE III

Expected tuberculosis morbidity in the State of New York

AGE GROUP	TUBERCULOSIS DEATH RATES PER 100,000 AS OF JULY 1, 1930 (U. S. BUREAU OF THE CENSUS)				MORTALITY PER 100,000 OF A POPULATION CONSISTING OF FIVE-SIXTHS WHITES AND ONE-SIXTH NONWHITES		ESTIMATED MORBIDITY PER 100,000		TOTAL MORBIDITY EXPECTATION FOR GENERAL POPULATION		
	White male	White female	Non-white male	Non-white female	Five-sixths white	One-sixth non-white	1/10 white	1/3 non-white	Corrected rate of reference	Uncorrected	Corrected
15–19	9.71	11.88	206.57	247.03	18	76	180	240	50,000	—	—
20–24	22.97	35.48	322.48	269.86	48	99	480	693	50,000	—	—
25–29	28.62	32.67	181.96	259.68	51	74	510	367	50,000	—	—
30–34	43.08	25.11	336.59	194.65	57	89	570	443	100,000	—	—
35–44	66.94	21.80	404.48	106.44	74	85	740	425	100,000	—	—
45–54	120.14	26.29	351.35	197.25	122	91	1,220	456	100,000	—	—
55–64	160.33	24.11	293.26	57.65	103	89	1,030	292	100,000	—	—
Total..	52.32	21.85	244.76	160.70	473	573	4,730	2,856	550,000	1.08	1.37

TABLE IV. *Tuberculosis morbidity rates in the twin index families*

	GENERAL POPULATION* OVER FOURTEEN YEARS OF AGE	RELATION TO TUBERCULOUS INDEX CASES					
		Husbands and wives	Parents	Half-siblings	Full siblings	Dizygotic cotwins	Monozygotic cotwins
Number of cases							
Cases of tuberculosis	—	14	114	4	136	42	48
Rates of reference							
Uncorrected	—	226	688	42†	720†	230	78
Corrected	—	197.5	676	33.5	534	164	55
Morbidity rates							
Uncorrected for differences in age	1.08	6.2	16.6	9.5	18.9	18.3	61.5
Corrected (Weinberg method)	1.37	7.1	16.9	11.9	25.5	25.6	87.3

* Estimated for a population in which the ratio of white to nonwhite persons approximates 5:1.

† All individuals above the age of fourteen.

TABLE V. *Average age, exposure and family taint of the index cases*

TYPE OF TWINS	AGE AT DISEASE ONSET		EXPOSURE OF INDEX CASES (PER CENT)		FAMILY HISTORY OF TUBERCULOSIS OF NONEXPOSED INDEX CASES (PER CENT)		
	Number of index cases	Average age	Definitely exposed index cases	Index cases with questionable exposure	Non-exposed index cases	Index cases with tuberculosis in direct ancestry	Tuberculosis in collateral lines only	No history of tuberculosis in ancestry
		years						
Monozygotic	78	21.3	35.9	11.5	52.6	36.6	22.0	41.5
Dizygotic	230	21.8	32.6	12.2	55.2	13.4	26.8	59.6
Total number	308	21.5	33.4	12.0	54.5	19.0	25.6	55.4

TABLE VI *Variations in sibship morbidity according to sex and parental taint*

	CORRECTED MORBIDITY RATES IN RELATION TO PARENTAL TAINT			CORRECTED MORBIDITY RATES IN RELATION TO SEX					
				Same-sexed sibships			Opposite-sexed sibships		
	Sibships with nontuberculous parents	Sibships with tuberculous parents	Total morbidity rate	Male	Female	All same-sexed persons	Male	Female	All opposite-sexed persons
Siblings	18.1	35.3	25.5	24.7	29.5	27.7	19.7	24.4	21.7
Dizygotic cotwins	18.8	38.6	25.6	27.6	31.6	30.2	18.6	22.7	20.5
Monozygotic cotwins	85.7	88.9	87.3	76.9	93.3	87.3	—	—	—

TABLE VII *Age variations and time of observation in the twins and siblings*

	ALL TUBERCULOUS PERSONS	AVERAGE AGE AT DISEASE ONSET (YEARS)			NONTUBERCULOUS COTWINS AT THE TIME OF EXAMINATION		AVERAGE DURATION OF OBSERVATION (YEARS)	
		Male	Female	Total average age	Total number	Average age	Observed concordance in concordant twin pairs	Observed discordance in discordant twin pairs
				years		years		
Siblings	136	27.9	22.5	24.3	—	—	—	—
Dizygotic cotwins	42	29.3	23.8	26.8	188	30.3	5.0	6.2
Monozygotic cotwins	48	26.5	23.9	25.3	30	30.9	3.5	5.7
Total number	*226	27.8	23.1	24.8	218	30.4	4.2	6.1

TABLE VIII

Distribution of the sibship morbidity and fatality rates and their variations according to race and the course of disease in the index cases

	HALF-SIBLINGS	ALL FULL SIBLINGS	SIBLINGS OF ALL DECEASED TWINS			ALL COTWINS	
			White	Nonwhite	Total	Dizygotic	Monozygotic
Number of persons							
All fatal tuberculosis cases	2	72	20	8	28	22	23
Corrected rates of reference	33.5	534	185	35	220	164	55
Corrected rates (Weinberg method)							
Fatality	5.97	13.5	10.8	22.9	12.7	13.4	41.8
Nonfatal morbidity	5.97	12.0	13.9	3.4	12.6	12.2	45.5
Total corrected morbidity	11.9	25.5	24.6	26.3	25.2	25.6	87.3

TABLE IX *Distribution of exposure in the concordant and discordant twin pairs*

TYPE OF TWINS	COTWINS WITH KNOWN EXPOSURE			COTWINS WITHOUT KNOWN EXPOSURE			ALL COTWINS		
	All exposed cotwins	Concordant	Discordant	All non exposed cotwins	Concordant	Discordant	Total number	Rate of cotwins in per cent	
		Number of persons	Per cent		Number of persons	Per cent			Concordant without exposure	Discordant with exposure
Monozygotic	52	36	69.2	26	16	61.5	78	20.5	20.5
			30.8			38.5			
Dizygotic	175	46	26.3	55	7	12.7	230	3.0	56.1
		129	73.7		48	87.3			
Total number	227	82	36.1	81	23	28.4	308	7.5	47.1
		145	63.9		58	71.6			

TABLE X

Variations in resistance to tuberculosis as indicated by the differences in extent and course of the disease between the two members of twin pairs

CLASSIFICATION OF SIMILARITY IN BEHAVIOR TO TUBERCULOSIS	EXTENT AND COURSE OF TUBERCULOSIS IN TWIN PAIRS				NUMBER OF TWIN PAIRS	
	First member		Second member		Monozygotic	Dizygotic
	Clinical classification	Subgroup	Clinical classification	Subgroup		
Complete dissimilarity	Fatal	IV	No tuberculosis (exposure)	I	0	38
	Fatal	IV	No tuberculosis (no exposure)	Ia	0	7
Less complete dissimilarity	Fatal	IV	Minimal with arrest	II	1	16
	Advanced	III	No tuberculosis (exposure or no exposure)	I, Ia	8	86
Complete similarity	Minimal with arrest	II	Minimal with arrest	II	3	2
	Advanced	III	Advanced	III	8	3
	Fatal	IV	Fatal	IV	9	7
Less complete similarity	Minimal with arrest	II	No tuberculosis (exposure or no exposure)	I, Ia	22	57
	Advanced	III	Minimal	II	14	8
	Fatal	IV	Advanced	III	13	6
Total number of pairs						
All dissimilar pairs					9	147
All similar pairs					69	83
Grand total					78	230
Ratio						
No tuberculosis to fatal tuberculosis					0:78	1:4
Dissimilar resistance to similar resistance					2:16	2:1

TABLE XI

Rates of satisfactory resistance to tuberculosis in the twin partners of tuberculous index cases

CLASSIFICATION OF SATISFACTORY RESISTANCE	TYPE OF TWINS	
	Monozygotic	Dizygotic
Total twins at risk	78	230
Favorable resistance of cotwins		
Number of pairs with complete and less complete dissimilarity in behavior to tuberculosis	9	147
Rate per 100 population	11.5	63.9
Very favorable resistance of cotwins		
Number of pairs with complete dissimilarity in behavior to tuberculosis	0	45
Rate per 100 population	0	19.5

and clinics in the State and City of New York, which reported a total number of 637 twin cases within a period of about five years. The criteria for selection of the index cases were that they be born by multiple birth and that they had come under observation of one of the cooperating agencies with the diagnosis of tuberculosis.

3. The material used for analysis of the statistical data on manifest reinfection tuberculosis consists of 308 complete twin families with 616 twin partners, 930 full siblings, 74 half-siblings, 688 parents, and 226 marriage partners of twin patients. The random sampling of these 308 twin pairs is indicated by the proportion of 78 monozygotic to 230 dizygotic pairs, a ratio of about 1:3.

4. Consistency in the calculation of collective morbidity figures for the various sibship groups was obtained by the use of the Abridged Weinberg Method.

5. Comparison of the corrected morbidity rates reveals that the chance of developing tuberculosis increases in strict proportion to the degree of blood relationship to a tuberculous index case. The difference in morbidity between dizygotic and monozygotic twin partners amounts to a ratio of 1:3.5.

6. The difference between the dizygotic and monozygotic co-twins increases to a ratio of 1:16, if the similarities in extent, course and eventual outcome of the disease are taken as additional criteria of comparison.

7. The consistent differences in tuberculosis morbidity among the various sibship groups of the twin index cases cannot be adequately explained on the basis of a simple correlation between closeness of blood relationship and increasing similarity in environment.

8. The analysis of the morbidity distribution in the sibship groups indicates that resistance to tuberculosis is modified by a heredo-constitutional mechanism which seems to be multi-factorial in its genetic nature.

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BACKCROSS PROGENY

Figure 8

Eight of the 117 offspring from the mating of the F_1 females back to the pure Duroc-Jersey male. *A*—A cherry red individual; *B*—Typical red; *C*—Light sandy; *D*—Red with a good belt; *E* and *F*—Two blacks with typical belts; *G*—Black pig lacking the belt and the domniant white on the legs; *H*—Black individual of one of the belted genotypes but having the belt expressed only on the right side. *D* and *H* are the cross-over types, wherein belting and black color are not associated.

LINKED COLOR FACTORS IN HAMPSHIRE SWINE

Linkage of Black and the Basic White of the White Belt Pattern

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THE white belt pattern in the Hampshire breed of swine has been known to be complicated in its inheritance. Although several papers have been published on the subject, the author knows of no analysis that has been made with the aid of the data from a test-cross generation. Spillman[13] reported that even amongst registered Hampshires about ten per cent of the progeny are without a belt. He concluded that the inheritance of the belt pattern depends on two or more factors. Lloyd-Jones and Evvard[5] have found that the belted pattern in swine is genetically complex. Olbrycht[9] analyzed the results of crosses of black and belted in the Wessex Saddleback breed of swine and found that the all-blacks differed from each other in their genotypes. Frőlich,[2] Simpson,[11] and Lloyd-Jones[5] have shown that the black of the Hampshire is dominant to the red of the Duroc-Jersey or Tamworth breeds.

Smith, Robinson and Bryant[12] have recently reviewed the literature of color inheritance in the pig. Earlier reviews are given by Wright,[16] Wentworth and Lush,[15] and Kosswig and Ossent.[3,4]

The cross of Hampshire males on Duroc-Jersey sows is often used for the production of market hogs. This cross was made at my father's (Frank J. Bushnell's) farm at Big Rock, Illinois. Having the F_1 material available, we decided that the F_1 Hampshire-Duroc-Jersey females with typical belts should be mated to a pure Duroc-Jersey boar in order to test the mode of inheritance of the various color factors involved.

Results

One of the two original Duroc-Jersey sows used in this experiment was of a dark sandy type (Figure 9*A*) and the other was red. The P_1 Hampshire male was black with a very wide belt. The seventeen offspring were black with varying amounts of white. Twelve had complete belts varying from a small band of white in some individuals to belts of about half the width of that of the P_1 male in others (Figure 9*B*). In no case did the belt at all approach the extent of the belt exhibited by the P_1 male. The remaining five F_1 individuals were black with one or more white forelegs.

The F_1 females selected for the production of the test-cross generation all had belts, narrow in three and of medium width in the others. The Duroc-Jersey male to which these six F_1 females were mated was a typically red individual of good breed type.

The test-cross generation of 117 individuals was classified according to color and as to the presence or absence of the white belt. The non-belted contingent was divided into two groups as follows: (1) those with one or both forelegs white (later considered as possessing the basic white of the belt pattern); and (2), those of solid color. The results of the classification are as follows: 37 entirely red or sandy; 24 entirely black; 8 red or sandy with one or both forelegs white; 15 black with one or both forelegs white. In the belted group, 16 were red or sandy and belted; and 17 black individuals were completely belted.

There were 61 individuals that exhibited the red or sandy color and 56 that were either black or black with some white. This indicates that the red (or sandy) and black segregated in the test-cross in the proportion of nearly 1:1.

If we now consider that the white is a dominant factor and that its presence is necessary for a belt to develop, we can

PARENT AND HYBRID TYPES

Figure 9

A—One of the P_1 Duroc-Jersey females of a dark sandy type. *B*—One of the F_1 females with the typical hampshire coloration of black with a white belt over the shoulders. This female has the widest belt of any of the F_1 individuals but hers is only about one-third the width of that of the P_1 male.

examine the data for the distribution of white in the test-cross. There were 56 individuals that got some white and 61 that were self colored. This indicates that white has segregated as though it were determined by a single factor.

Considering now only the groups that had white forelegs or a complete belt, it will be noted that the completely belted contingent comprises 33 of a total of 56 that obtained the basic white factor. According to the interpretation given by Spillman[13] where two factor pairs are considered necessary to extend the belt over the shoulders, we would expect only a quarter to have complete belts and three-quarters to fall in the partially belted classification. This would be 14 belted and 42 with one or both forelegs white, whereas 33 and 23 were the numbers obtained. If only one factor pair is involved in the extension of the white of the forelegs over the shoulders to form the belt, we would expect 28 belted and 28 partially belted. The one-factor extension hypothesis appears to explain the results more satisfactorily.

It is noteworthy that individuals lacking white on both forelegs never developed white on the shoulders. This would indicate that the second belting factor involved is truly an extension factor for the white that occurs on the forelegs.

If duplicate factors were involved in the development of the belted condition we would expect 29 belted, 58 partially belted, and 29 non-belted (no white) in a test cross. We actually obtained 33 belted, 23 partially belted, and 61 non-belted which is obviously not a good fit.

Assuming that the pigs having the belt and those having one or both white front feet have received the basic white factor of the belting complex, we can re-examine the data for the distribution of that white gene. In the test-cross the results are as follows: 37 self red and 32 black and white (composed of the belted and white foreleg contingents) representing the original combinations; and 24 self black and 24 red belted or partially belted representing the new combinations. This would indicate a partial linkage of black color and the basic white of the belt complex with a white of the belt complex with a cross-over value of 41.02 per cent ($n = 117$).

In order to further test the significance of the assumption that black and the dominant white of the belting complex are linked, the per cent of cross-over for the 1941 and the 1942 crops of pigs was calculated for each year. In the 1941 group the cross-over value was 40.91 per cent ($n = 66$) and in the 1942 group it was 41.17 per cent ($n = 51$). The results for the two years are in significant accord.

So far, all pigs not black have been referred to as red or sandy. One of the

BLACK BACKCROSS PROGENY

Figure 10

Four black individuals showing variations in the expressivity of the belted pattern. *A*—is the typical with white fore-legs and a complete (extended) belt. *B*—has the dominant white on the legs but lacks the extension factor which completes the belt. *C*—has the dominant white and the extension factor but modifiers have restricted the white to a small patch on top of the shoulders. *D*—a female of the belted genotype but showing a non-germinal variation limiting the belt to the left side.

P_1 females was sandy and the other was red. Of the 1942 pigs, the 23 non-black individuals were red or sandy in the proportion of 14 and 9. The Duroc-Jersey male used to produce the test-cross generation must have been heterozygous for one of the sandy genes. The sandy pigs of the 1942 crop were in the proportions four sandy belted (including partially belted) and five self sandy.

Discussion

The above results clearly indicate that the basic white concerned in the development of the white belt pattern of the Hampshire breed is dominant. One main extension factor is necessary for the white to be extended from the forelegs over the shoulders. The width of the belt (effectiveness of extension of the white) probably depends on several pairs of modifying factors (Figure 10*A, B, C, D*). Selection within the Hampshire breed seems to have been toward a belt of medium width but there is within the breed sufficient heterogeneity to allow for wide variations in belt size. The selection within the Duroc-Jersey breed seems to have been for those modifying factors which tend to restrict the development of the complete belt as well as for the complete suppression of white on the body. Although the P_1 Hampshire male had a very wide belt, the factors transmitted by the P_1 Duroc-Jersey females were capable of restricting considerably the development of the belt in the offspring. It would seem to follow that had a Hampshire male of a narrow belt type been used, even fewer of the offspring would have had complete belts.

Some strains of Duroc-Jerseys may, however, carry white factors which become evident with inbreeding. Nordby[8] reported for certain inbred Duroc-Jerseys 68 individuals marked with white (including one with a white belt and one with an incomplete belt) among a total of 196 pigs. The white factor (or fac-

tors) present in such Duroc-Jerseys would appear to be recessive and capable of being increased in amount by elimination of modifiers which tend to restrict this white. In some cases it is possible for this recessive white to be extended over the shoulders in the form of a belt, indicating that all strains of the breed are not lacking in the belt extension factor. In the Duroc-Jersey breed selection has been in the direction of elimination of white, a process that can apparently be accomplished both by the elimination of dominant white and by selecting modifiers which restrict recessive and perhaps dominant white. The same thing seems to have happened in the Tamworth breed as McPhee[7] has found the belting tendency present in inbred families of that breed. Actually, the same white may be carried by Hampshires on the one hand and by the Duroc-Jerseys and Tamworths on the other, and we are dealing simply with a question of varying dominance. The expressivity of white in Durocs may have been lowered by reason of the selection of modifiers which make it appear to behave as though it were recessive. In the Hampshires, modifiers have been selected which enhance the production of white to the point that white appears to behave as a dominant.

Considerable variation was noted in the width of the belt, or in its absence, the amount of white on the forelegs (Figure 10). That some of this variation is non-germinal is indicated by the belt width differences that occur on the two sides of the body. Also the leg of one side may be white and the other black in individuals where the belt is absent. Two animals were noted that had a perfect belt on the left side of the body but none on the right side, the belt being interrupted at the mid-dorsal line. Another pig had a perfect belt only on the right side (Figure 8*H*). Wright and Chase[17] have found that the amount of white in guinea pigs is influenced by a multiplicity of genes with individually small effects and also that there is an enormous amount of non-genetic variability which is responsible for a wide range in the amount of white. It would appear reasonable to conclude that a similar situation is involved in the development of white patterns in domestic swine. It must be kept in mind, however, that the white factor being influenced in the Hampshire and other belted breeds is primarily a dominant one.

Wentworth and Lush[15] have shown that two factors are involved in the development of the typical red color of Duroc-Jerseys. These factors are cumulative in action and one may act as an intensifier of the other, the absence of either one resulting in sandy. The results of the present investigation suggest that the Hampshire lacks at least one of these red-producing genes. The test-cross generation produced several sandy individuals some of which were quite light in color (Figure 8*C*). and others with perfect belts.

Summary

A test-cross of hybrid Duroc-Jersey-Hampshire females mated with a Duroc boar indicated that the belting complex of the Hampshire is dependent on a basic white factor and one main extension factor for that white. There are probably minor factors which either aid or restrict the activities of the major extension factor, and some non-germinal variation.

The basic white factor of the belting complex is dominant and appears to be linked with the dominant gene which produces the black coat. The cross-over between the basic white and the black genes is approximately 41 per cent.

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GENETICS AND THE SYSTEMATIST

THERE have been numerous books on the "species question," but no others based on quite the background of this one.* Dr. Mayr is a taxonomist, and (as the title indicates) this is the chief basis of his discussion. However, he also has a wide acquaintance with the literature of Genetics,† being much more at home in this field than a taxonomist usually is. The book is organized as one might expect of a taxonomist: as each new topic is taken up, there comes first a logical classification of its contents, and then a discussion that closely follows this classification. The result of this system is that the arguments are clearly presented, easy to follow, and easy to look up afterwards.

The account is limited to animals, but with this limitation there is an attempt at a general synthesis. Mayr is himself an ornithologist, and he recognizes the difficulty of coming to a real understanding of the systematics of unfamiliar groups, but has nevertheless believed it worth trying. He will not be surprised that many specialists in other groups will object to his treatment of their material. The present reviewer, for example, disagrees with a number of the interpretations concerning ants and Drosophila—the two groups with which he is most familiar.

There are innumerable examples cited in the book, mostly from birds and mostly from those of the South Seas. The result is that the great majority are unfamiliar, and concern forms that are mere Latin names—at least to the present reviewer. It is, therefore, in most cases not possible to form any independent judgment as to how far the arguments from these examples may be trusted. The use of birds for the main part of the argument is justified by the author, on the grounds both of his more intimate knowledge of them, and of the fact that the group is the most nearly completely known among all animals. Both points are well taken—though they do not make matters any easier for a reviewer with little knowledge of ornithology. However, one may well question the statement that "more than 98 per cent of all species [of birds] probably existing are known and described." It would seem that this is a matter on which adequate quantitative data cannot be obtained.

According to Mayr, the tendency in ornithology has been to reduce species to subspecies, so that the total number of distinct species recognized has decreased while the number of named forms (subspecies) was increasing. He foresees a similar process occurring in other groups as they become better known, and believes that reasonable

MAYR, E. Systematics and the Origin of Species. Pp. XIV+334. Columbia University Press. 1942.

†In at least one instance there is an unfortunate quotation from this literature. The example is not important in the present case, but the reviewer was disappointed to see the reference to the mutational change in temperature tolerance in Daphnia. This case has been cited several times recently in genetical literature; examination of the original account will suggest that the evidence is not strong enough to bear the arguments based on it (by other authors, not by Mayr). My own guess is that no mutation was involved, but only an accidental and temporary change in the fauna or flora of the culture medium.

stability in nomenclature will arise elsewhere as it has with birds. In many individual cases the decision between specific or subspecific status for a given form is largely a matter of taste, but general agreement can usually be reached. To the reviewer it seems probable that in many cases this general agreement is an arbitrary one. This does not decrease its usefulness to the cataloguer, to the student of ecology, of distribution, or to any one else who has to use names. It does raise doubts as to how far one is to trust a smoothly working nomenclatorial system when it comes to arguments concerning evolution.

Mayr's defines a species as follows:

A species consists of a group of populations which replace each other geographically or ecologically and of which the neighboring ones intergrade or interbreed whenever they are in contact or which are potentially capable of doing so (with one or more of the populations) in those cases where contact is prevented by geographical or ecological barriers.

Or shorter: species are groups of actually or potentially interbreeding natural populations, which are reproductively isolated from other such groups.

This definition admittedly involves a considerable element of personal judgment in its application to particular examples—but this is unavoidable on any workable definition. The reviewer is inclined to agree that a species is usually a real natural phenomenon, even though its delimitation is often difficult.

Mayr insists that the criterion of interbreeding must be studied under natural conditions. The fact that two forms will interbreed in captivity is not to be taken as conclusive evidence that they do so under natural conditions. The absence or rarity of intermediates in the field is the critical evidence. While the point is well taken, the reviewer would also insist that neither field observations nor laboratory experiments can exclude the possibility of occasional crossings which may be of great importance as making available a whole series of new genes.

The general conclusion reached is that practically all specific diversity arises through geographical isolation in the first instance. In many cases two closely related species have overlapping geographical ranges, but these are supposed to be secondary, due to migration after the origin of reproductive isolation. While such a supposition is not unreasonable, it does make the general scheme difficult to check—for almost any imaginable combination of geographical ranges now becomes consistent with the theory. This type of argument occurs repeatedly in the book—for example in connection with multiple invasions of islands by successive migrants from a common stock. The conclusions are consistent with the data, but are not thereby proven to be correct, since no attempt is made to try out other types of interpretation.

In general, the geographical basis for the origin of interspecific reproductive isolation seems the most probable one. But this is chiefly because it is difficult to imagine the working of any alternative. To the reviewer it seems that Mayr has underestimated ecological isolation (he wonders if a more detailed knowledge of ants or of plants might not modify Mayr's views here), and the possibilities of rapid change in small temporary subpopulations. There are certain logical difficulties in picturing the development of reproductive isolation as a result of geographical separation. Given two strains, at first fully cross fertile, and now geographically separated; it is supposed that they will gradually diverge, and that such divergence will entail some degree of cross-sterility. But this does not avoid the essential difficulty in the origin of cross-sterility; for each successive step, if it result in some degree of sterility, will be selected against. We are thus forced to picture the postulated gradual evolution of cross-sterility as proceeding in spite of a selection against each new stage as soon as it arises. The only escape from this conclusion is through the hypothesis of complementary genes. On this basis a new gene does not lead to cross-sterility except when combined with another gene which exists only in the other population. Some such assumption seems to be necessary in any case, since the necessity is not avoided by postulating geographical isolation.

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AGRONOMIC SMUT

JOHN W. TAYLOR AND HARRY V. HARLAN*

THE layman has long been confused over the working of the Scientific Mind. The usual concept is one of men with knee-sprung trousers selecting bottles from endless rows and pouring the contents together. One comes out with gunpowder and another with a salve for poison ivy. All is the result of Profound Concentration. Chance never enters the picture except where Something Boils Over. That is always good. In this paper the writers have attempted by honest reporting to reveal both the mental processes of man and the bebehavior of loose smut in barley.

The writers, despite their well-known pathological leanings, did not start out to perform a pathological experiment. Moreover, had not the Junior Author been afflicted with senility, the results would have been agronomic. It happens that at Arlington, Va., a variety known as Nakano Wase has been grown for many years. During these years it has been conspicuous for the large number of F_1 hybrids which appeared annually. The Junior Author suggested that this was probably due to the fact that flowering glumes of the lateral florets in this variety are interlocked but lightly and they, therefore, stand open at flowering time. It was decided that two plantings be made at Aberdeen, Idaho, from seed grown at Arlington. One planting was to consist of central kernels, the other of lateral kernels from the same spikes. The Senior Author sent the seed as planned, Mr. Stevens, at Aberdeen, planted it as directed, but the Junior Author forgot what it was about and took notes on smut instead of hybrids. Since the smut happened to be loose smut, the original thesis was not affected, for the lateral flowers still stood open.

In Table I it will be seen that the incidence of smut was much greater in the lateral flowers. This was true not only in Nakano Wase, but also in Esaw. Esaw is a hybrid derived from Nakano Wase.

Incidentally, neither Nakano Wase nor Esaw is susceptible to the local "nigra" smut. The percentages were not calculated in 1935, but each row was seeded with approximately the same number of seed. The results were so striking that by 1937 it was decided to repeat the experiment at Arlington, Va. The results confirmed those of 1935. Three selections were used and in each of these the percentage of smut was much greater in the plants from lateral kernels than in those from central kernels.

In 1938 a refinement was added by the Senior Author. In addition to the usual lateral and central kernel experiments which, incidentally, gave results in line with those of 1935 and 1937, he conceived of a labor-saving device of using screens of a suitable mesh to separate the central from the lateral kernels. While this is not wholly exact, it was accurate enough to yield results parallel with those already obtained. As can be seen in the table no smut was found in plants from very large kernels, while 17.5% was found in the plants from small ones. In 1939 the seed was separated with a Hero fanning mill. The screens were not as suitable as those used the previous year, but the separation was rapid and simple and physically less exhausting. It will be noted that the Nakano Wase results in Table II are in about the usual percentages. In varieties such as Nakano Wase the benefits of the fanning mill go much farther than had been previously claimed. It not only removed kernels smutted by cereal smut, but eliminates many of those that are going to have

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loose smut the following year.

Very little smut was found in Wisconsin Winter. The lemma and palet of the lateral kernels are interlocked or overlapped in this variety. The slight difference between the infection in plants from large and small kernels in Wisconsin Winter clears up one point not previously discussed. The lateral flowers mature later than the central, which are often in the boot at flowering-time. This in itself allows greater opportunity for infection in the lateral flowers but apparently this difference is slight except in such varieties as Nakano Wase where the lemma and palet stand open at flowering time.

It might also be mentioned that a rechecking of the 1935 material showed a greater number of natural hybrids from the lateral flowers, the actual data being 19 to 5. The authors find the formulation of conclusions a bit difficult. The knowledge that loose smut does take advantage of the opportunity for infection afforded by the open lateral flowers of certain varieties may aid in our breeding program. At the same time it is heartening to know that truth too persistently evident may be come upon in various ways, and if absentmindedness is a firm basis for original work greater things may be expected soon.

TABLE I. Number and percent of loose smut heads (*Ustilago nuda*) from central and lateral kernels seeded separately.

Place	Year	Variety	Total Heads		Smutted Heads		Percent Smutted Heads	
			Central Kernels	Lateral Kernels	Central Kernels	Lateral Kernels	Central Kernels	Lateral Kernels
Aberdeen, Idaho	1935	Esaw			9	72		
		Nakano Wase 68			4	96		
Arlington, Va.	1937	Esaw	1294	2082	20	156	1.5	7.5
		Nakano Wase 51	1754	2429	106	495	6.0	20.4
		Nakano Wase 33	1723	2351	99	250	5.7	10.6
Arlington, Va.	1938	Nakano Wase 68	1439	1301	40	196	2.8	15.1

TABLE II. Number and percent of smutted heads (*Ustilago nuda*) from barley seeds of two varieties separated in size groups by screens and fanning mill at Arlington, Va.

Year	Variety	Method of Separation	Size of Seed	Size of Screen	Total Heads	Smutted Heads	Percent Heads Smutted
1938	Nakano Wase 68	Tyler Screen	Small	Thru No. 8	819	143	17.5
			Medium	Thru No. 6	2301	215	9.3
			Large	Over No. 6	413	0	0
1939	Nakano Wase 68	Hero fanning mill	Small	Thru 12 x 2.3	1388	272	19.6
			Small	Thru 12 x 2.5	911	158	17.3
			Medium	Over 12 x 2.5	1408	87	6.2
			Large	Over 6 x 1.5	1185	65	5.5
	Wisconsin Winter	do	Small	Thru 12 x 2.3	535	14	2.6
			Small	Thru 12 x 2.5	1494	47	3.1
			Medium	Over 12 x 2.5	1629	22	1.4

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Bumper Baby-Crop in 1943

Without the propaganda and pressure which have characterized the intensive campaigns of the Axis countries for more babies, the birth rate in the United States has been moving upward quite steadily from the minimum point (16.6) it had reached in 1933. * * * By 1942 it had jumped to 20.9 per 1,000. When the accounts are closed for 1943, the indications are that the rate may reach about 24 per 1,000, the level prevailing during the first World War. Because of the increase in population during the past quarter of a century, the actual number of babies born in 1943 will far exceed that for any previous year in the country's history.—*Statis. Bull. Metro. Life Ins. Co.*

INHERITANCE OF A CYANOGENETIC GLUCOSIDE AND ITS HYDROLYZING ENZYME IN *TRIFOLIUM REPENS*

S. S. Atwood and J. T. Sullivan†

With most species of plants that produce hydrocyanic acid (HCN) there is danger of poisoning when sufficient plant material is ingested by ruminant stock. In the case of white clover, however, no fatalities or evidences of toxicity have ever been reported. The reason for this different behavior of white clover is not directly related to the amount of HCN, since it can be shown that the HCN consumed under some grazing practices with white clover may be several times the quantity estimated as lethal. Further feeding trials are needed where the amount of both the glucoside which contains HCN and the enzyme which liberates the HCN can be controlled.

The first report of the presence of a cyanogenetic glucoside in white clover and of an enzyme necessary for its hydrolysis was made by Mirande[13] in 1912, but no studies on the inheritance of HCN were reported before those of Williams[17] in 1939. Williams found monohybrid ratios regularly in backcross, F_2 and F_3 generations, and he postulated a single recessive factor, *ac*, to explain the acyanogenetic condition.

It was noted by Armstrong, et al[1] in 1913 and by Pethybridge[14] in 1919 that HCN was found more regularly in English wild type plants than in plants from commercial sources. The principal application of this knowledge has been made in New Zealand, where Doak[9] showed that the better white clover strains contained more glucoside than did the more poorly adapted strains. Recently, Foy and Hyde[10] applied the glucoside test to seed certification, since it was found that the types giving the higher tests were superior in regard to such characters as total production, seasonal distribution, and persistence under competition. Williams[17] showed, on the other hand, that this relationship did not hold for over 30,000 individuals collected from a number of sources.

The particular glucosides reacting in the picric acid test have been isolated by Melville and Doak[12] and shown to be lotaustralin and linamarin. The enzyme capable of hydrolyzing these compounds was found by Coop[7] to be identical with the linamarase prepared from linseed. Using purified preparations of these compounds, Corkill[8] found some plants containing enzyme alone, some glucoside alone, some both, and some neither.

Materials and Methods

The sources of the two inbred lines used in this study were respectively a seed collection in an old pasture in West Virginia and a vegetative isolation from a closely grazed pasture on the Pennsylvania State College farms. All crosses were made in the greenhouse using methods previously reported.[3]

The picric acid test for glucoside was used essentially as decribed by others.[15,17] At first the ten leaves picked from each plant were frozen and toluene was added before enclosing the moistened paper strip, but in later tests the freezing was omitted with no apparent difference in results. When enzyme or glucoside solutions were added to the samples, thorough mixing was effected by grinding the contents of each tube together with a small quantity of pure quartz sand.

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†Associate Geneticist and Physiologist, respectively.

Since difficulty was encountered in obtaining a sufficient quantity of crystallized glucoside for so many tests, plant juice was used in its place with satisfactory results. This juice was obtained by pressing leaves which had been autoclaved to inactivate the enzyme. The final extract, after clarification, was a clear liquid, shown to possess a specific amount of glucoside but no enzyme activity.

Experimental Results

Results with Standard Picric Acid Test

In the first cross studied the picric test was used without the addition of glucoside or enzyme; i.e., it was essentially the control test of Corkill.[8] The female parent of this cross was selected from a second-generation inbred progeny derived by self-pollination. The original parent of the inbred line gave a negative reaction to the picric acid test (indicating no glucoside present); each of its 90 F_1 inbred progeny tested 0, and each of the 49 second generation inbreds tested 0. The male parent was selected from a third generation inbred line that had consistently tested positive for HCN throughout the three years of inbreeding. The F_1 plants were grown in the greenhouse, where they were intercrossed and backcrossed by hand. All 25 F_1 plants tested positive for HCN in the greenhouse and in the field after transplanting. The seed for the F_2 and backcross (B) generations were germinated in the greenhouse, and the young plants were transplanted to the field as spaced individuals. After about four months' growth, the plants were sampled for HCN by removing ten young leaves of approximately the same age from the periphery of each plant.

Although considerable variation was obtained in the degree of darkening of the picric acid test strip, in general the reaction could be easily classified either as positive or as no reaction. When the tests were graded after about 18 hours reacting time, many showed no perceptible change from control and were classified as "0." Others gave a very slight color change, which was often of questionable significance. These latter reactions were classified as grade 1 and for summary purposes were included with the 0 group. Increasingly darker shades of reddish-brown were classified as groups 2, 3, or 4, and some tests which showed almost a dark purple color were classed as 5. In summarizing, all distinctly positive testing plants, namely those of classes 2, 3, 4, and 5, were added together. Of the 607 F_2 plants, 225 tested 0, 31 tested 1, 7 tested 2, 33 tested 3, 228 tested 4, and 83 tested 5. This frequency distribution was distinctly bimodal and lends support to the separation of "positive" and "negative" plants at the point between grades 1 and 2. Likewise among the 499 backcross plants, 272 tested 0, 23 tested 1, 6 tested 2, and 38 tested 3, 124 tested 4, and 36 tested 5. Whenever individual plants were retested in the same year, in different years, or in greenhouse and field, approximately the same degree of color reaction was obtained. It appeared from all the data that the different amounts of glucoside, as indicated by the different amounts of color, were not due to fortuitous circumstances, but were conditioned genetically by factors modifying the influence of the main factors which were segregating for presence or absence of the glucoside. No attempt was made, however, to study the hereditary behavior of the modifying genes.

The F_2 population consisted of 23 families derived by crossing 16 F_1 plants in various combinations. Using the X^2 to test for goodness of fit to a 3:1 ratio, it was found that 11 of the 23 F_2 families differed significantly from expectation, all except one being deficient in plants testing positive (Table I). Likewise both the total X^2 and X_t^2 for 3:1 exceeded a P of .01. It appeared that this population was not segregating uniformly in a monohybrid ratio. On the other hand, with most of the families and with the total F_2 population, X^2 showed a good fit to a 9:7 ratio, expected with two complementary factors.*

*The values of Chi-square were corrected for continuity wherever necessary according to the suggestions of Cochran.[6]

By inspection it was apparent that certain families fitted a 3:1 ratio better than a 9:7. Such a situation would be possible if one parent had been homozygous for both dominant factors while the other parent had been heterozygous for one factor and homozygous recessive for the other. On this basis, two equal classes of F_1 plants would be expected: one class heterozygous for both factors; the other class heterozygous for one factor and homozygous dominant for the other. If plants of the former class were intercrossed, 9:7 ratios would be expected in the F_2, whereas any intercrosses involving the latter class of F_1 plants would be expected to give 3:1 ratios in the F_2. Since most of the F_1 plants were involved in more than one intercross and nine of them were backcrossed to their negative parent, all ratios involving any one F_1 plant were compared to determine whether the plants were heterozygous for both or for only one factor. In this way, 12 plants were classed as heterozygous for both factors, one appeared heterozygous for one, whereas the other three plants could not be classified definitely on the basis of the X^2 test. If these latter three were classed according to their more probable X^2, two appeared to be heterozygous for both factors, and one heterozygous for one, making a total of 14:2, a ratio that differs significantly from the expected 1:1. This is a small population, however, and since all other results in the *B*, F_2 and F_3 generations fit the hypothesis as outlined, it is considered a satisfactory explanation.

On the basis of two types of F_1 plants, giving respectively two types of ratios in the F_2, it is evident that the F_2 families segregating in the two ways should be considered separately rather than together. When the totals for the four families segregating for one factor were added, total X^2, X_t^2 and X_h^2 showed a good fit to 3:1; three of these families showed a deficiency of positive testing plants when compared with expectation on a 3:1 hypothesis, while the fourth was an exact fit to expected. The same four families did not fit a 9:7 ratio as shown by X_t^2. With the 19 families segregating for two factors, total X^2, X_t^2 and X_h^2 indicated a satisfactory fit to 9:7; ten of these families showed a deficiency of positive testing plants when compared with the expected number based on a 9:7 ratio, and nine of them had an excess. In only two of the families did X^2 exceed the value for $P = .30$. These same 19 families did not fit a 3:1 ratio as evidenced by the highly significant total X^2 and X_t^2.

Confirmatory results were obtained in the backcross population, obtained by mating nine F_1 plants with the parent that had given a negative HCN test. X^2 for three of the *B* families, total X^2, X^2_t and X_h^2 for the total *B* population, showed an unsatisfactory fit to 1:1 (Table II), indicating that the single factor hypothesis[17] did not hold. Likewise when fitted to a 1:3 ratio, the values of X^2 for three families, total X^2, X_t^2, and X_h^2 showed that the two-factor hypothesis did not apply if the acyanogenic parent was homozygous recessive. On the other hand, the hypothesis that two factors were involved and that the negative parent was homozygous recessive for one factor and heterozygous for the other, gives a satisfactory fit to expectation. One of the F_1 plants that had given a 3:1 ratio in the F_2 was included among the plants that were backcrossed. The resultant backcross family (No. 30, Table II) gave a satisfactory fit to 1:1, and the X^2 test indicated that the same family did not fit either a 1:3 or a 3:5 ratio. Seven of the other families were descended from F_1 plants shown by the F_2 test to be heterozygous for two factors, while the eighth family came from a plant not included among the parents of the F_2. Six of these families did fit a 3:5 ratio satisfactorily (three of them to the exclusion of the 1:1 ratio), but the other two gave significant values of X^2 for 3:5. One of them, however, only slightly exceeded the value for $P = .05$, and the other consisted of only 21 plants, possibly an inadequate sample. In addition, the total X^2, X_t^2, and X_h^2 for these eight families all showed satisfactory fit to 3:5.

Because the F_2 and *B* results had been different from Williams',[17] an F_3 generation was tested. It was obtained by backcrossing 21 positively reacting F_2 plants with their original negative parent. According to the hypothesis accepted for the F_2 and *B*, four types of families were expected: Those testing uniformly positive, those segregating 3:1, those segregating 1:1, and those segregating 3:5. It was found that one family of 30 plants consisted of only HCN+ individuals, whereas all others segregated (Table III.) In order to classify them into the three segregating types, the families were grouped according to their lowest X^2. Six families gave a good fit to only one of the three ratios while the X^2 value for the other two ratios was significant. Eleven other families showed a significant X^2 for only one of the ratios, and the remaining three families had none of the X^2 values significant. The most important feature of these results seems to be the fact that all four expected types of families were obtained and that among each of the four types at least one family occurred to the exclusion of all other types.

The expected frequency of the four family types in F_3 depends on whether the F_2 plants used as parents occurred in a 9:7 progeny or in a 3:1 progeny. The former should be in the ratio 1:2:2:4, while the latter should be 1:1:2:2 (since the latter all resulted from crossing plants heterozygous for one factor with plants heterozygous for both rather than from crossing two plants each heterozygous for the same factor) (Table I). Seventeen of the 21 F_2 plants that were tested were of the former type, and the other four were the latter making a theoretical ratio for the 21 plants of 2.6:4.4:5.1:8.9. This approximates very closely the obtained ratio of 1:4:6:10, the X^2 value being only 1.286, a non-significant value.

Interactions of Glucoside and Enzyme

At this stage in the study the work of Melville and Doak[12] and of Coop[7] was published describing in white clover the specific glucosides and the enzyme responsible for the positive picric acid test. Corkill[8] at about the same time described plants with only one or the other constituent as well as plants with both and plants with neither. This suggested the possibility that one of the two factors reported here conditions glucoside irrespective of the enzyme, while the other conditions enzyme irrespective of the glucoside. Since most of the plants tested previously had been destroyed, it was thought desirable to begin with a new cross for a study of the enzyme and glucoside separately.

Among the plants studied, the only one known definitely to be homozygous for both factors was the one which had given 100 per cent HCN positive progeny when backcrossed to its original HCN negative parent (Table III). This plant had been increased vegetatively in the field and was chosen as the male parent of the new cross.

In selecting the HCN negative parent an attempt was made to prepare the glucoside and enzyme according to the directions of Melville and Doak[12] and of Coop.[7] Since difficulty was encountered in obtaining the crystallized glucoside, the concentrated extract was used directly in liquid form to test for plants with enzyme but no glucoside. The four types of plants described by Corkill[8] were distinguished in the same manner that he described.*

Several F_3 plants which had given a negative control test also gave no reaction when glucoside or enzyme was added. A vigorous representative was chosen from this group to be used as the female parent for the new cross. When this same plant was tested subsequently in the greenhouse using purified plant

***Differentiating Tests for Enzyme and Glucoside.**

Plants with	*Control test*	*Enzyme added*	*Glucoside added*
Both enzyme and glucoside	+	+	+
Enzyme but no glucoside	0	0	+
Glucoside but no enzyme	0	+	0
Neither glucoside nor enzyme	0	0	0

juice as the source of the added glucoside, it tested 0 0 +. This suggested the possibility of variation in enzyme action under different conditions, but all later results agreed with the second test indicating that the plant did have enzyme. In the tests for selection of the 0 0 0 parent none of the plants testing 0 0 + gave as dark a test with glucoside added as other plants gave in the control. Apparently the first glucoside solution was not sufficiently concentrated or purified, and on this account an error was made in classifying the parent. Since the new F_2 and *B* populations were already growing by this time, the study was continued on the chance that segregation might be obtained for both characters.

Because it had been noted previously that individual plants could be tested for HCN more economically by growing in individual pots in the greenhouse than by transplanting to the field, all F_1, F_2, and *B* plants from the second cross were handled in this way. All 26 F_1 plants tested + + +; 13 of them were used for intercrosses, selfings, or backcrosses.

Since the backcross population was expected to yield the most critical evidence as to the type of segregation, it was tested first (Table IV). If the presence of glucoside and of enzyme were each conditioned by single factors, and if the female, 0 0 + testing parent, were heterozygous for the enzyme factor, two types of F_1 plants would be expected with equal frequency: Those heterozygous for both factors, and those heterozygous only for the glucoside factor. When the former were backcrossed to their female parent, a 3:3:1:1 ratio was expected; when the latter were backcrossed, a 1:1 segregation only for glucoside was expected. Ten of the 13 families segregated for both enzyme and glucoside whereas the other three families segregated only for glucoside. This 10:3 segregation does not differ significantly from that expected on a 1:1 hypothesis.

In four backcross families the total deviation from expected was significant. By calculating values of X^2 for the separate factors and for linkage, it was seen that in every family and in the total population the segregation for enzyme gave a satisfactory fit to 3:1. On the other hand three families differed significantly from the expected 1:1 ratio in their segregation for glucoside. Since the deviations involved both excesses and deficiencies of the positive-testing plants in different families, both total X^2 and X_h^2 for the glucoside segregation were significant. These discrepancies, however, tended to cancel one another, so that the X_t^2 was not significant. It was seen on closer examination that the direction of the deviations was correlated with the way in which the backcross was made. When the female parent (34-I) was used as male in the backcross, all five families had an excess of HCN+ plants. When the female parent was used as female, on the contrary, six of the eight families had a deficiency of HCN + plants, one had a slight excess, and the other exactly fitted expectation. If these two groups are considered separately, not only total X^2 but X_t^2 for each group is highly significant. It appears at first that the diverse ratios might be conditioned by differential pollen tube growth and that the glucoside factor might, therefore, be linked to the oppositional alleles conditioning cross-incompatibility.[2] No such explanation will hold, however, since the female parent was homozygous recessive for the glucoside factor. The cause of these aberrant glucoside ratios is being investigated further.

The one other *B* family with significant total X^2 had a significant X^2 for linkage. Since the X^2 values for linkage in the total population were non-significant, however, the significance of linkage in the backcross is questionable. Except for the three aberrant glucoside segregations, all evidence favors a dihybrid ratio for the two factors responsible respectively for glucoside and enzyme.

Similar evidence was obtained from the F_2. In this case a large population had been started, but only those families were tested in detail in which segre-

gation for both factors was expected on the basis of the *B* tests of the F_1 plants. Two of the families were derived by crossing plants heterozygous for both factors, and the other four came from self-pollination. Since no significant differences were found between these two types of families, only the totals for the entire population are presented (Table V). One family had a significant total X^2, which could be attributed mainly to a significant deviation in enzyme segregation. This value only slightly exceeded the value for *P* of .05, however, and the values of X^2 for the total enzyme segregation were non-significant, probably indicating a satisfactory agreement with 3:1. In these F_2 families, glucoside segregation likewise fitted a 3:1 ratio, three of the families having slight excesses of HCN+ plants and the other three having deficiencies. Here as in the *B* one family had a significant X^2 for linkage. In addition three other families exceeded the value of X^2 for $P = .30$, so that the total X^2 for linkage also was significant. Two families had an excess of HCN+ plants in the parental combinations, and three others had a deficiency. Because of this, X_h^2 was significant, but the deviations tended to cancel one another, thus making X_t^2 not significant. It should be noted that the one family with significant X^2 for linkage as well as the total population showed a deficiency rather than an excess of original combinations, thus eliminating the possibility of linkage. When all the *B* and F_2 evidence is brought together, it gives little indication of linkage between the factors for glucoside and enzyme.

Certain of the F_2 and *B* families showing segregations differing significantly for both enzyme and glucoside were later retested. Additional progenies were grown in an attempt to determine more accurately the cause of the aberrancies. Plant 34-11 (Table V) was selfed again in the greenhouse, and the 77 additional progeny which were obtained segregated 44:12:14:7. Fitted to a 9:3:3:1 ratio, the corrected X^2 values for enzyme, glucoside, and linkage were respectively 0.108, 0.004, and 1.052, none of which was significant. Likewise, when 298 additional plants were grown from the *B* families obtained from plants 34-11, 34-12, 34-13, and 34-14 (Table IV), the segregations for enzyme, glucoside, and linkage fitted the expected 3:3:1:1 ratio for each of the families and for the total population (119:95:40:44). With these same families, none of the values of total X^2, X_t^2, or X_h^2 was large enough to be significant. All these data support the hypothesis of single unlinked genes determining respectively the presence of glucoside and of enzyme.

On the other hand, the results were more complicated when the same four plants (34-11, 34-12, 34-13, and 34-14) were crossed with two individuals which had tested 0-0-0 and therefore were presumed to be homozygous recessive for both the enzyme and glucoside factors. From these crosses, 1:1:1:1 ratios were expected, but discrepancies were obtained in several cases. When plant 34-11 was crossed with one of the tester plants, the resultant ratio of 15:6:21:20 yielded a significant corrected X^2 (5.823) for enzyme segregation. In addition each of the four families derived from this first tester had a deficiency of recombination plants, so that the total segregation for the four families (78:57:67:94) gave a highly significant corrected X_t^2 (7.463) for linkage. With the second tester plant, the segregation obtained from plant 34-12 (35:17:18:12) showed significant corrected X^2 for enzyme (5.378) and glucoside (6.451) segregation. Likewise, the segregations from plant 34-13 (35:14:8:13) gave highly significant corrected X^2 for enzyme segregation (10.414) and for linkage (8.929). Both total X^2 and X_t^2 for enzyme, glucoside, and linkage were significant with the four families from the second tester. To obtain these ratios, reciprocal crosses had been added together, since it had been observed in all previous work that reciprocals were not significantly different. In this case, however, when the tester was used as female, all families fitted the expected 1:1:1:1 ratio except that from 34-13

(17:9:4:11), which gave a significant corrected X^2 (4.780) for linkage. On the other hand when the tester was used as male, significant corrected values of X^2 for enzyme were obtained with three families and for glucoside with two families.

Although most of the results with the two additional tester plants confirm the original hypothesis, there is also evidence that some linkage may exist between the factors, and that modifying genes may have a pronounced influence on the segregation.

Mention should also be made of the variation observed in amount of both glucoside and enzyme when they were tested for separately. In general the test for glucoside alone tended to give a darker reaction than that for enzyme alone, but this could be altered simply by changing the concentrations of the stock solutions used. Variations between plants using the same solutions were just as conspicuous. For example some plants tested 1-4-1 or 0-5-0 whereas most of the plants classed as having glucoside but no enzyme tested 0-4-0. Likewise other plants tested 1-1-4, indicating a very slight amount of glucoside and a large amount of enzyme. These intermediate tests were usually obtained the second time when certain plants were retested. It appears that both glucoside and enzyme may vary in amount, so that various amounts of either or both would account for the differences in the control test. Some families showed consistently more variation than others in this respect, indicating a heritable basis for it, but no simple genetic mechanism was evident among the plants tested.

Relation with Other Plant Characters

Segregation for the normal V-shaped white marking on the leaf blades also occurred in part of the F_2 population. Both parents and all F_1 plants showed the dominant marking, but since segregation occurred in some F_2 families, it is evident that one of the parents and certain F_1 plants must have been heterozygous for this factor. Three families consisting of a total of 97 plants were classifiable, and the segregation for marking was considered separately in combination with the glucoside and with the enzyme segregation. The segregation for leaf marking (74:23) approximated 3:1 very closely, and in neither case was there evidence of linkage.

The segregating populations classified here offered favorable material for testing the correlation between HCN and good vegetative growth such as was found by Doak[9] and other workers in New Zealand. The 607 F_2 plants and the 499 *B* plants examined in the first cross, plus 90 F_2 plants from a sister cross showing the same type of segregation, were noted for vegetative vigor at the end of their first summer's growth in the field. All plants were classed as 1, 2, 3, 4, or 5, representing increasing degrees of vigor as shown by their spread, height, and density. The correlation coefficient between these vigor ratings and the HCN ratings for the same plants was —0.035, a non-significant value. The presence of a severe rust epidemic and the prevalence of virus during the following summer made classification for vigor or for persistency less meaningful at this time. In addition when several hundred inbred lines were noted in the field, no relation was observed between their vigor ratings and the amount of HCN. Apparently with breeding material from sources other than New Zealand, a correlation between vegetative vigor and HCN may not be the rule.

Discussion

Of primary interest in connection with the present study is the fact that there have been identified genes which have a direct effect on a specific enzyme and on a specific chemical constituent of the plant. Demonstrations of such cases of direct gene action are relatively infrequent.

Perhaps of equal interest, however, is the complementary relation between these genes. If only the control test were made, the phenotypic effect of the two separate genes was not evident, resulting in a 9:7 ratio. This alone could be interpreted to mean that both genes

TABLE I. Segregations in F_2 for presence (+) and absence (o) of glucoside as indicated by the picric acid test.

No.	F_2 families Origin	No. plants testing +	No. plants testing o	Total plants	Corrected X^2 for 3:1†	9:7†
1	15-1 x 15-3	21	11	32	+ 1.041	+ .793
2	15-1 x 15-13	16	9	25	+ 1.080	+ .336
3	15-2 x 15-13	13	11	24	+ 4.500 *	+ .000
4	15-3 x 15-13	18	9	27	+ .805	+ .805
5	15-5 x 15-13	14	19	33	+ 16.979 **	+ 2.032
6	15-6 x 15-13	16	13	29	+ 5.068 *	+ .005
7	15-7 x 15-13	22	12	34	+ 1.412	+ .674
8	15-8 x 15-13	16	12	28	+ 3.857 *	+ .907
9	15-9 x 15-1	12	6	18	+ .326	+ .417
10	15-9 x 15-2	9	12	21	+ 9.920 **	+ 1.035
11	15-9 x 15-13	18	16	34	+ 7.887 **	+ .046
12	15-10 x 15-1	20	11	31	+ 1.301	+ .558
13	15-10 x 15-13	10	13	23	+ 10.565 **	+ 1.049
14	15-12 x 15-1	11	7	18	+ 1.185	+ .032
15	15-13 x 15-1	13	7	20	+ .600	+ .318
16	15-13 x 15-12	16	14	30	+ 6.400 *	+ .019
17	15-14 x 15-1	12	9	21	+ 2.893	+ .081
18	15-14 x 15-13	21	7	28	o .048	+ 3.275
19	15-15 x 15-1	14	5	19	+ .017	+ 1.692
20	15-15 x 15-13	24	12	36	+ .925	+ 1.193
21	15-16 x 15-1	4	9	13	- 11.308 **	- 2.475
22	15-19 x 15-1	12	14	26	+ 10.061 **	+ .706
23	15-19 x 15-13	19	18	37	+ 9.811 **	+ .189
	Total F_2	381	286	667		
			Total X^2		127.467 **	28.028
			X^2_h		- 94.577 **	+ .580
			X^2_n		32.176	25.414
	6 families segregating 3:1 (Nos. 17, 18, 19 & 20)	71	33	104		
			Total X^2		4.922	7.941
			X^2_h		+ 2.167	+ 5.627*
			X^2_n		2.409	1.836
	19 families segregating 9:7 (All except Nos. 17, 18, 19 & 20)	260	223	503		
			Total X^2		122.742 **	18.085
			X^2_h		- 99.251 **	.048
			X^2_n		22.466	18.016

* Significant - exceeds X^2 for P of .05
** Highly significant - exceeds X^2 for P of .01
† The + sign before the X^2 value indicates an excess of the positive testing plants; the - sign indicates a deficiency; and o indicates an exact fit to expected.

TABLE II. Segregations in backcross (B) families for presence (+) and absence (o) of glucoside as indicated by the picric acid test.

No.	B families Origin	No. plants testing +	No. plants testing o	Total plants	Corrected X^2 for 1:3†	1:1†	3:5†
24	15-1 x 15-I	54	61	115	+ 28.409 **	- .314	+ 3.994 *
25	15-2 x 15-I	4	8	12	+ .111	- .750	- .000
26	15-3 x 15-I	35	76	110	+ 2.376	- 13.828 **	- 1.283
27	15-4 x 15-I	11	18	29	+ 1.942 *	- 1.242	+ .021
28	15-9 x 15-I	26	47	73	+ 3.840	- 8.480 *	- .046
29	15-13 x 15-I	13	8	21	+ 13.349 **	+ .762	+ 4.346 *
30	15-14 x 15-I	29	20	49	+ 28.741 **	+ 1.306	+ 8.922 **
31	15-19 x 15-I	31	19	50	+ 1.805	+ 1.861	- .068
32	15-19 x 15-I	21	39	60	+ 2.689	- 4.816 *	- .017
	Total B	204	295	499			
			Total X^2		90.881 **	34.404 **	21.438 *
			X^2_h		+ 68.283 **	- 16.232 **	+ 2.293
			X^2_n		23.756 **	17.808 *	19.003 *
	8 families segregating 1:3 (All except 30)	175	275	450			
			Total X^2		80.344 **	32.750 **	11.808
			X^2_h		+ 65.559 **	- 21.780 **	+ .314
			X^2_n		14.048	10.528	11.238

* Significant - exceeds X^2 for P of .05
** Highly significant - exceeds X^2 for P of .01
† The + sign before the X^2 value indicates an excess of the positive testing plants; the - sign indicates a deficiency; and o indicates an exact fit to expected.

TABLE III. Classification of F_3 families according to most probable X^2.

Significance of X^2	Families not segregating	Families segregating 3:1	Families segregating 1:1	Families segregating 1:3	Total families
Other two ratios significant	1	2	1	3	7
One other ratio significant	-	2	2	7	11
None significant	-	-	3	-	3
Total obtained	1	4	6	10	21
Total expected	2.6	4.4	5.1	8.9	

X^2 = 1.286 3 D/P P = .95-.50

TABLE IV. Segregations in backcross (B) families for both enzyme and glucoside.

No.	B families Origin	No. plants with: Both enzyme and glucoside	Enzyme but no glucoside	Glucoside but no enzyme	Neither enzyme nor glucoside	Total plants	X^2 for: Enzyme segregation† (corrected)	Glucoside segregation† (corrected)	Linkage† (corrected)	Total
54	34-I x 34-3	2	7	2	1	12	o .111	- .750	- 1.361	3.111
56	34-I x 34-4	11	13	6	4	33	- .010	- .000	- .162	.374
56	34-5 x 34-I	14	12	0	0	26	--	- .019	--	--
57	34-6 x 34-I	22	15	0	0	37	--	+ .496	--	--
58	34-I x 34-7	10	12	3	2	27	+ .309	- .000	- .198	.951
59	34-I x 34-8	5	10	4	7	26	- 3.282	- 1.885	- .115	6.821
60	34-I x 34-9	17	15	0	0	32	--	- .016	--	--
61	34-I x 34-10	7	13	3	8	31	- 1.301	- 3.226	- .688	6.591
62	34-11 x 34-I	23	14	1	9	47	+ .177	+ .000	+ 7.262 **	8.092 *
63	34-I x 34-12	7	22	0	8	37	+ .081	- 13.081 **	- .577	15.252 **
64	34-13 x 34-I	25	6	4	3	38	+ .861	+ 9.500 **	+ 1.974	12.549 **
65	34-14 x 34-I	26	11	6	2	45	+ .896	+ 7.200 **	+ .030	9.361 *
66	34-I x 34-15	14	14	4	4	36	+ .037	o .028	o .009	.148
	10 families segregating for both enzyme and glucoside (All except Nos. 56, 57 & 60)	135	122	32	48	332				
						Total X^2	9.517	40.631 **	14.182	64.330 **
						X^2_h	+ .100	- .148	+ 3.037	3.486
						X^2_n	9.373	40.438 **	11.033	60.844 **
	3 families segregating only for glucoside (Nos. 56, 57 & 60)	53	42	0	0	95				
						Total X^2	--	.801	--	--
						X^2_h	--	.526	--	--
						X^2_n	--	.164	--	--

* Significant - exceeds X^2 for P of .05
** Highly significant - exceeds X^2 for P of .01
† For the enzyme and glucoside segregation, the signs before the X^2 values indicate relation of positive testing plants to expected; for the test of linkage, the signs indicate the relation of the original combinations to expected.

TABLE V. Segregations in F_2 for both glucoside and enzyme

No.	F_2 families Origin	No. plants with: Both enzyme and glucoside	Enzyme but no glucoside	Glucoside but no enzyme	Neither enzyme nor glucoside	Total plants	X^2 for: Enzyme segregation† (corrected)	Glucoside segregation† (corrected)	Linkage† (corrected)	Total
67	34-11 x 34-7	18	8	1	4	31	+ .871	- 2.419	+ 2.240	7.022
68	34-13 x 34-7	24	8	7	1	40	+ .300	+ .033	o .278	1.066
69	34-4 ⊕	14	6	11	0	31	- 1.301	+ .289	- 4.391 *	7.281
70	34-10 ⊕	14	12	5	1	32	.375	- 3.375	- 2.347	7.866
71	34-11 ⊕	17	1	9	4	31	- 3.882 *	+ .871	+ 1.881	7.939 *
72	34-15 ⊕	14	7	3	0	24	+ 1.389	- .056	- .907	3.407
	Total F_2	101	42	36	10	189				
						Total X^2	11.080	9.486	13.723 *	34.241 *
						X^2_h	+ .016	- .510	- .986	1.768
						X^2_n	11.016	8.821	12.636 *	32.473 **

* Significant - exceeds X^2 for P of .05
** Highly significant - exceeds X^2 for P of .01
† For the enzyme and glucoside segregation, the signs before the X^2 values indicate relation of positive testing plants to expected; for the test of linkage, the signs indicate the relation of the original combinations to expected.

had a direct effect on the production of a cyanogenetic glucoside in the plant. By the addition of the prepared enzyme and glucoside to the tests, however, it was seen that only one of the genes had a direct effect on glucoside production whereas the other one was concerned only with the production of enzyme. It is possible that other characters that are influenced by more than one gene may be controlled by a similar mechanism. For example Stadler[16] has speculated that certain of the genes affecting plant color in corn may condition enzyme formation, whereas others are known to affect more directly the type of molecule involved. Likewise with the many, often complementary, genes conditioning flower color, it is suggested[11] that enzyme action may be genetically controlled. If the end products of other gene actions could be analyzed into their constituent parts as they were here by the use of the purified glucoside and enzyme, it seems reasonable to suppose that certain complex ratios might be analyzed more completely into a direct effect of several genes, the total effect producing a single phenotypic character.

From the standpoint of chemistry there is one feature of this analysis that is hard to reconcile with present knowledge of enzyme action. It is often assumed that an enzyme responsible for the decomposition of a compound is also responsible for its synthesis. This would mean in the case of the cyanogenetic glucoside studied here that an enzyme capable of releasing HCN for the picric test might also condition the glucoside's synthesis under other conditions. If this assumption in regard to enzyme action is correct, it is difficult to see how plants could contain glucoside but no enzyme. An explanation may perhaps be found in the fact that the picric test is only roughly quantitative, and that the plants testing 0 + 0 really have a slight amount of enzyme, adequate for synthesis of the glucoside but too little to be detected by the picric test.

In many ways it is a rather remarkable biological phenomenon that in New Zealand HCN is generally confined to the better adapted plants of white clover. Since the plant is not native to that region, it is conceivable that such a system might arise when the species was first introduced, but it is more difficult to think of a selection mechanism that would maintain such a relation after the species had been widely cultivated over a number of years. In most species the characters of yield and persistence have been found to be conditioned by at least several genes, and white clover appears to be no exception. It does not seem probable that a simple linkage exists between the genes affecting the presence of glucoside and of enzyme and those affecting yield and persistency. It would appear more likely that the first plants introduced into New Zealand happened to be homozygous for these HCN genes, and that mutations to those causing lack of enzyme or glucoside were rare and not of importance in a crop that was increased rapidly.

No linkage was definitely established between any of the three genes studied here, but this might be expected since the plant has 32 somatic chromosomes. Disomic inheritance was found for the multiple alleles conditioning cross-incompatibility[2] and for other factors conditioning certain leaf markings (unpublished). This together with the regular bivalent formation at meiosis[4] was interpreted as evidence that white clover is an allotetraploid—rather than an autotetraploid. The disomic segregation reported here supports that conclusion.

Summary

1. In a cross between two inbred lines of white clover respectively testing negative and positive for HCN by the picric acid test, the F_1 tested uniformly HCN positive; the F_2 families segregated in part 9:7 and in part 3:1, and the backcross families in part 3:5 and in part 1:1. This is interpreted to mean that the presence of HCN is conditioned by two complementary dominant factors and that one parent was homozygous for both factors while the other parent was homozygous for the recessive of the first factor and heterozygous for the second.

This hypothesis was confirmed when 21 HCN positive F_2 plants were backcrossed to their HCN negative parent.

2. In a second cross between a plant breeding true for HCN and another HCN negative plant, the tests for HCN were made by adding prepared solutions of glucoside and of enzyme separately for each plant tested. Segregations in both F_2 and backcross indicated that the presence of glucoside and of enzyme were each conditioned by a single dominant factor, corresponding to the two factors noted in the first cross. Most of the evidence indicated no linkage between these factors.

3. No linkage was observed between the factor for white marking on the leaf blade and the factors for either glucoside or enzyme.

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R. A. Fisher Appointed to Cambridge

It has been announced (*Nature*, August 7, 1943) that the chair of genetics in the University of Cambridge is to be filled by the appointment of Prof. R. A. Fisher, who has been Galton professor in the University of London since 1933. Before that time he was head of the Statistics Department at Rothamsted Experimental Station. Prof. Fisher has been actively associated with genetics since 1918 when he showed that the biometrical correlations observed between relatives were to be expected on the Mendelian theory. He has written extensively on the action of selection, and he has shown that the manifestation of any gene must be regarded as modifiable through the action of selection on other genes in the nucleus. This culminated in his well-known theory of the evolution of dominance by selection and in his analysis of the genetical basis of mimicry and natural polymorphism. The mathematical theory of selection in relation to variation and mutation was further developed in *The Genetical Theory of Natural Selection* (1930), which also deals with the genetical aspects of human society. As would be expected, Prof. Fisher has been responsible for many improvements in the methods of coping with the peculiar difficulties of human data. His early work on the genetical interpretation and statistical analysis of biometrical data has also been extended, notably in a paper published jointly with F. R. Immer and O. Tedin in 1932. This work is perhaps less widely appreciated, largely because geneticists as a body have been shy of attacking the inheritance of quantitative characters; but interest in this subject is reviving, and Prof. Fisher's findings must have their influence on future developments here, as in other fields. It is indeed singularly appropriate that he should both have succeeded Pearson in London and become professor of genetics in the University where Bateson and his associates did so much to extend and amplify Mendel's results, for no one has played a larger part in uniting the once conflicting sciences of biometry and genetics.

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